WHAT IS CLAIMED IS:

Sub A1

1. A method of evaluating the atherosclerotic state of an individual, comprising the steps of:

(a) collecting tissue of interest from said individual;

(b) determining the amount of mitochondrial DNA damage in said tissue of interest; and

(c) comparing the amount of mitochondrial DNA damage in tissue of interest from said individual to the amount of mitochondrial DNA damage in tissue of interest from a control individual who does not have atherosclerosis, wherein a greater amount of mitochondrial DNA damage in said individual at risk than in said control individual is indicative of atherosclerosis in said individual.

2. The method of claim 1, wherein said mitochondrial DNA damage is determined by quantitative PCR.

3. The method of claim 1, wherein said individual has at least one risk factor associated with atherosclerosis.

4. The method of claim 3, wherein said risk factor is selected from the group consisting of tobacco smoking, hypertension, diabetes, obesity, hypercholesterolemia and hyperlipedemia.

5. The method of claim 1, wherein said mitochondrial DNA damage is assessed by a measurement selected from the group consisting of measurement of mitochondrial mRNA production, measurement of mitochondrial protein production, measurement of changes in mitochondrial oxidative phosphorylation and measurement of changes in mitochondrial ATP production

6. A method of measuring the amount of oxidative stress in an individual, comprising the steps of:

(a) collecting tissue of interest from said individual;

(b) measuring the amount of mitochondrial DNA damage in said tissue of interest;

(c) determining the amount of DNA damage in a nuclear gene in said tissue of interest; and

(d) comparing the amount of DNA damage per length of DNA between said mitochondrial DNA and said nuclear gene, wherein a greater amount of mitochondrial DNA damage per length of DNA than nuclear DNA damage per length of DNA is indicative of an increased amount of oxidative stress in said individual.

7. The method of claim 6, wherein said nuclear gene is selected from the group consisting of the β-globin locus, transcriptionally active genes, and transcriptionally inactive genes.

8. The method of claim 6, wherein said mitochondrial DNA damage and DNA damage to said nuclear gene is determined by quantitative PCR.

9. The method of claim 6, wherein increased amounts of oxidative stress are predictive of atherogenesis, hypertension, diabetes mellitis, hypercholesterolemia, cigarette smoking, degenerative diseases of aging and cancer.

10. The method of claim 6, wherein said mitochondrial DNA damage is assessed by a measurement selected from the group

consisting of measurement of mitochondrial mRNA production, measurement of mitochondrial protein production, measurement of changes in mitochondrial oxidative phosphorylation and measurement of changes in mitochondrial ATP production

Sub A2

11. A method of determining the efficacy of a drug to reduce the risk of atherosclerosis in an individual, comprising the steps of:

(a) collecting tissue of interest from said individual prior to and subsequent to administering said drug to said individual;

(b) determining the amount of mitochondrial DNA damage in said tissue of interest collected, wherein a decrease in mitochondrial DNA damage subsequent to said treatment is indicative of a treatment that reduces the risk of atherosclerosis.

12. The method of claim 11, wherein said mitochondrial DNA damage is determined by quantitative PCR.

13. The method of claim 11, wherein said mitochondrial DNA damage is assessed by a measurement selected from the group consisting of measurement of mitochondrial mRNA production, measurement of mitochondrial protein production, measurement of changes in mitochondrial oxidative phosphorylation and measurement of changes in mitochondrial ATP production.